

## Correspondence

### Epidermal antibodies in secondary syphilis

TO THE EDITOR, *British Journal of Venereal Diseases*

Sir,

Syphilitic patients may show symptoms of an 'aberrant' antibody synthesis. Antibodies bearing evidence of this are rheumatoid factor and antibodies against erythrocytes. Wright *et al.* (1970) have found that sera of patients with early syphilis contain antibodies, which stain cell mitochondria in a fluorescent test. These antibodies are not organ-specific and are characterised by their absorption with Venereal Disease Research Laboratory antigen, from which cardiolipin was shown to be the active ingredient.

When looking for autoantibodies in sera of patients with early syphilis (less than two years), we found fluorescence with several tissue substrates which could also be absorbed with cardiolipin.

The sera from 26 patients with untreated syphilis were tested by the indirect immunofluorescence technique (IFT) for the presence of autoantibodies. Six of these 26 patients had seronegative primary syphilis, six serolabile primary syphilis, two seropositive primary syphilis, seven secondary syphilis, and five early latent syphilis. The serological subdivision of primary syphilis was made by applying antilipoidal and antitreponemal tests (Kolmer, VDRL, Reiter protein complement-fixation, and *Treponema pallidum* immobilisation): seronegative primary syphilis was diagnosed when all results were negative; serolabile primary syphilis when some were positive; and seropositive primary syphilis when all were positive. The tissue substrates used for the IFT for autoantibody determination were unfixed, frozen, human parotid, adrenal, and toxic thyroid glands and rat heart, stomach, striated muscle, and kidney. Guinea-pig lip was used as substrate for skin-antibodies.

We found that:

1. The sera from all the patients with seropositive primary or secondary syphilis produced a strong granular fluorescence of

guinea-pig lip. This fluorescence differed from the pattern regularly found with the sera from patients with pemphigus or parapemphigus.

2. The same sera also produced a strong smooth muscle fluorescence, which was different from the pattern that is observed with sera in active chronic hepatitis or primary biliary cirrhosis.

The sera from patients with seropositive primary or secondary syphilis also caused weak fluorescence with striated muscle and heart muscle and with parotid and thyroid glands.

The fluorescence in epidermis, smooth muscle, striated muscle, heart muscle, parotid and thyroid glands could be abolished by preabsorption of the sera with cardiolipin. Control absorption with lecithin and phosphate buffered saline was negative. Treatment of the patients with penicillin led to a decrease in the titre of the antibodies.

In the sera from patients with seronegative or serolabile primary syphilis and in the sera from three of five patients with early latent syphilis no such antibodies could be found with any of the substrates tested, although occasionally doubtful fluorescence of smooth muscle, epidermis, striated muscle, heart muscle, and thyroid gland was seen. In the two other cases of early latent syphilis the sera showed fluorescence of smooth muscle and epidermis, whereas fluorescence with other substrates was weak or absent.

Unlike Wright *et al.* (1970) we did not find a pattern of mitochondrial fluorescence in the distal tubules of kidney. This may be caused by the fact that we used rat kidney instead of human kidney. Although with rat kidney mitochondrial fluorescence in sera from patients with syphilis has been described (André *et al.*, 1973). As nearly all types of fluorescence could be absorbed with cardiolipin we found with the indirect immunofluorescence technique no indications that an infection with *Treponema pallidum* induces the synthesis of antibodies against several autoantigens as suggested by

Allison (1973) and recently by Primi *et al.* (1977). These findings confirm those of Doniach (1976).

Yours faithfully,

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### References

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### Tetracycline treatment for non-specific urethritis

TO THE EDITOR, *British Journal of Venereal Diseases*.

Sir,

Dr Fowler (1978) disagrees with my advocacy of a long follow-up for the assessment of treatment of non-specific urethritis, stating that it 'was shown to be unnecessary in a study (Fowler, 1970) in which the follow-up was not the one year Mr Simopoulos finds so admirable, but three years, and the long-term findings proved of no help in assessing treatment'. In saying this, Fowler is doing scant justice to his own paper. He then refers to a time 'in the past when the aetiology